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Post-traumatic Stress Disorder Moderates the Relation Between Documented Childhood Victimization and Pain 30 Years Later

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Abstract

Cross-sectional designs and self-reports of maltreatment characterize nearly all the literature on childhood abuse or neglect and pain in adulthood, limiting potential for causal inference. The current study describes a prospective follow up of a large cohort of individuals with court-documented early childhood abuse or neglect (n=458) and a demographically matched control sample (n=349) into middle adulthood (mean age 41), nearly 30 years later, comparing the groups for risk of adult pain complaints. We examine whether Post-Traumatic Stress Disorder (PTSD) mediates or moderates risk of pain. Assessed prospectively across multiple pain measures, physically and sexually abused and neglected individuals generally showed a significant ($p<.05$) but notably small (η^2 =.01) increased risk of pain symptoms in middle adulthood. Although PTSD was associated with both childhood victimization ($p<.01$) and risk of middle adulthood pain ($p<.001$), it did not appear to mediate the relationship between victimization and pain. However, across all pain outcomes other than medically unexplained pain, PTSD robustly interacted with documented childhood victimization to predict adult pain risk: Individuals with both childhood abuse/neglect and PTSD were at significantly increased risk ($p<.001$, η^2 generally = .05–.06) of pain. After accounting for the combined effect of the two factors, neither childhood victimization nor PTSD alone predicted pain risk. Findings support a view that clinical pain assessments should focus on PTSD rather than make broad inquiries into past history of childhood abuse or neglect.

Keywords

childhood trauma; childhood abuse; childhood neglect; post-traumatic stress disorder; unexplained pain symptoms

1. Introduction

Despite a growing body of research, the role of childhood abuse and neglect in development of pain in adulthood remains controversial. Some reviews strongly support the relationship [3,32,38]. Others [12,13,24] suggest that the relationship is modest at best. Problems

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plaguing nearly all research in this field include retrospective reporting of prior childhood events and cross-sectional designs, rendering interpretation of positive findings difficult [45].

In the last decade, reports from four prospective studies [9,18,26,28] have appeared. Only the last two [9,26] avoid reliance on self-report by using court records of child maltreatment to classify participants. Both found weak and nonsignificant relations between verified child abuse or neglect and adult pain, although results were in the predicted direction. Nevertheless, because the mean age of both cohorts was mid-to-late 20s, follow up into later life stages is critical.

One psychiatric diagnosis associated consistently with both pain [1,2,4,5,[21],25,30,33–37] and childhood victimization [44] is post-traumatic stress disorder (PTSD). We [22,24] and others [17,19] have contended that comorbid PTSD may be important in understanding the relationship between childhood victimization and adult pain.

PTSD may mediate or moderate the relationship between childhood victimization and adult pain. PTSD symptoms have been shown to mediate the relationship between self-reported childhood maltreatment [17] or self-reported childhood sexual abuse [10] and body pain. PTSD has also been shown to mediate other health outcomes of traumatic life events [15], and self-reported [40,48] and documented [42] child maltreatment. A mediational model suggests that trauma increases risk of PTSD and that PTSD is the proximal cause of ill health.

Alternatively, PTSD may moderate the relationship between PTSD and pain. An effect moderator model proposes that the relation between childhood trauma and pain is solely or more strongly found in individuals who meet PTSD criteria. A moderator model was supported in one study [41] reporting that psychiatrically-diagnosed PTSD plus a major trauma history increased risk of lifetime medical problems compared to those with a trauma history alone.

PTSD may be viewed as a marker of stress vulnerability [23,25], in which those susceptible to stress are more likely to develop chronic pain and other negative health consequences, when a stressful exposure occurs. A moderator model of PTSD, childhood victimization, and adult pain suggests that the childhood victimization and pain relationship is particularly marked among individuals with a PTSD history, but smaller or absent among those without PTSD.

The current investigation aims to (a) extend longitudinal examination of the relation between documented childhood victimization and pain into middle adulthood, and (b) examine whether a history of PTSD mediates or moderates the relationship between childhood victimization and pain in middle adulthood.

2. Methods

2.1 Design and Sample

Subjects utilized for these analyses are drawn from participants in a prospective cohort study in which abused and/or neglected children were matched with non-abused and non-neglected children of similar age, race/ethnicity, gender, and parental social class and followed prospectively into adulthood. Because of the matching procedure, the participants are assumed to differ only in the risk factor; that is, having experienced childhood physical or sexual abuse or neglect. Since it is not possible to assign subjects randomly to groups, the assumption of equivalency for the groups is an approximation. The control group may also

differ from the abused and neglected individuals on other variables associated with abuse or neglect. For complete details of the study design and subject selection criteria, see [43].

The original sample of abused and neglected children (N = 908) was made up of substantiated cases of childhood physical and sexual abuse and neglect processed from 1967 to 1971 in the county juvenile (family) or adult criminal courts of a Midwestern metropolitan area. Only court-documented cases of child abuse and neglect were included, thereby identifying a group of abused and/or neglected children whose cases came to the attention of authorities at that time. Abuse and neglect cases were restricted to those in which the children were less than 11 years of age at the time of the abuse or neglect incident.

A comparison cohort was matched as closely as possible on the basis of gender, age, race, and approximate parental socioeconomic status during the initial period under study (1967–1971). Matching was accomplished by dividing the cohort of abused and/or neglected cases into two groups according their age at the time of the abuse or neglect incident. Children who were younger than school age at the time of the court-documented incident were matched with children of the same gender, race, date of birth ([+ or −] 1 week), and hospital of birth through the use of county birth record information. For children of school age, records of more than 100 elementary schools for the same time period were used to find matches with children of the same gender, race, date of birth ([+ or −] 6 months), same class in same elementary school during the years 1967–1971, and home address (within a five-block radius of the abused or neglected child, if possible). Overall, there were 667 matches (74%) for the abused and neglected children.

The second phase involved tracing, locating, and interviewing study participants during 1989 through 1995 in *young adulthood*, an average of 22 years after their childhood maltreatment experiences. During this phase, participants were assessed across multiple domains of functioning, including cognitive, intellectual, emotional, psychiatric, social, and interpersonal. Interviewers were blind to the purpose of the study, to the participants' group membership, and to the inclusion of an abused and/or neglected group. Similarly, participants were blind to the purpose of the study; they were told they had been selected to participate as part of a large group of individuals who grew up in that area in the late 1960s and early 1970s. Institutional Review Board approval was obtained and participants completed an informed consent process, documenting that they understood the conditions of their participation and that they were participating voluntarily.

Of the original sample of 1,575, 1,307 subjects (83%) were located and 1,196 (76%) were interviewed in young adulthood (1989–1995). Of those not interviewed, 43 were deceased, 8 were incapable of being interviewed, 268 were not found, and 60 refused to participate. Comparison of the young adulthood follow-up sample with the original sample indicated no significant differences in terms of percentage male, white, abused and/or neglected, type of abuse or neglect, poverty in childhood census tract, or mean age at time of initial accrual. This young adulthood follow-up sample, with an average age of 29.1 years (SD=3.8), was the sample utilized in pain-related analyses reported earlier by Raphael et al. [26].

Another wave of follow-up interviews were conducted between the years 2003 and 2004. Compared to the first interview sample of 1,196 in young adulthood, 1,051 individuals (88%) were located and 807 (67%) were interviewed in 2003–2004. This *middle adulthood* sample includes 78 cases of physical abuse, 61 of sexual abuse, 370 cases of neglect and 349 matched controls. The total does not equal 807, because some participants experienced multiple types of victimization. Of those not interviewed, 47 were deceased, 6 were incapable of being interviewed, 145 were not found, and 190 refused to participate. Approximately half the middle adulthood follow-up sample was female (53%) and about

two thirds were white (63%). The mean age of the sample was 41.2 (SD=3.6) years. Only 59% of the sample had completed high school.

Comparison of the middle-adulthood interview sample with the young-adulthood interview sample indicated no differences in terms of percentage male, white, abused and/or neglected, type of abuse or neglect, poverty in childhood census tract, and mean age during young-adulthood interviews. The original and middle-adulthood samples also did not differ in terms of percentage male, white, abused and/or neglected (and physically or sexually abused), type of abuse or neglect, poverty in childhood census tract, and mean current age.

Full comparison of the young-adulthood and middle-adulthood samples, detailing both demographic measures and sources of attrition can be found in Bentley and Widom [7]

2.2 Measures and variables

2.2.1 Classification of abuse/neglect—Physical abuse cases included injuries including bruises, welts, burns, abrasions, lacerations, wounds, cuts, bone and skull fractures, and other evidence of physical injury. Sexual abuse charges ranged from relatively non-specific charges of "assault and battery with intent to gratify sexual desires" to more specific charges of "fondling or touching in an obscene manner," rape, sodomy, incest, etc. Neglect cases reflected a judgment that parents' deficiencies in childcare were beyond those found acceptable by community and professional standards at the time. These cases represented extreme failure to provide adequate food, clothing, shelter, and medical attention to children. Further details regarding subject selection criteria are available in Widom et al [43].

2.2.2 Pain—Identical measures of pain symptoms were collected during both young adulthood (1989–1995) and middle adulthood (2003–2004) interviews. Items from the Somatization module of the Diagnostic Interview Schedule III-R (DIS-III-R) [27] were used to assess the frequency of pain reports in young and middle adulthood. The number of pain symptom complaints was derived by summing the number of positive responses to questions about: Have you ever had abdominal or belly pain, back pain, pain in the joints, pain in arms or legs, chest pains, headaches, pain when you urinated, burning pain around private parts, and pain anywhere else. The following items were preceded by the phrase: "Have you ever had *a lot of trouble with*…": abdominal or belly pain, back pain, headaches. This measure is referred to as number of pain symptoms.

A second measure of pain symptoms was derived by subsetting the first symptom count, using further information derived from structured probes that are a standard part of the DIS-III-R. To determine the number of nontrivial pain symptoms, we summed number of pain symptoms to which there was a positive response and in which the respondent told the interviewer that they either told a doctor or other professional about the problem and/or reported that the symptom interfered with his or her life or activities a lot. This measure is referred to as number of pain problems. The next measure of pain symptoms was computed by summing the number of pain symptoms that the respondent indicated was always the result of a physical illness or injury. This comprised a measure of the number of pains attributed to illnesses/injuries.

The last measure of pain symptoms was derived by summing the number of pain symptoms that were experienced, but which were *not* attributed entirely to either medication, drugs, alcohol, physical illness or injury. This count of unexplained pain symptoms form a subset of the items that were typically used to render a DSM-III-R diagnosis of Somatization Disorder.

2.2.3 Post-Traumatic Stress Disorder (PTSD)—Lifetime history of PTSD was obtained from the National Institutes of Mental Health Diagnostic Interview Schedule (DIS), Version III-R [27] PTSD module, as conducted during young adulthood [44]. The DIS section on PTSD started with a question in which several typical PTSD events were mentioned, and respondents were asked whether any of these events had ever happened to them. The description of traumatic events followed the DSM-III-R text and used examples from that definition. A report of an event that did not fit the stressor definition (e.g., illness, divorce) was excluded from further inquiry, and the respondent was asked whether he or she had experienced another event of the sort described in the question. A respondent's report of a PTSD-type event was followed by questions about the occurrence of PTSD symptoms after the event. Up to three qualifying events were investigated regarding PTSD sequelae. "Lifetime" prevalence is the proportion of the group who ever experienced PTSD up to the time of the young adulthood interview (1989–1995). Note, however, that since lifetime PTSD was not assessed during middle adulthood, our assessment does not account for PTSD that might have been experienced between young and middle adulthood.

2.2.4 Control variables—*White, non-Hispanic* is a self-reported variable. Respondents who reported their race/ethnicity as white, non-Hispanic were coded as 1 (59% of the respondents were white, non-Hispanic). All others were coded as 0, with the largest remaining group compromised of black, non-Hispanic (34%).

Since subjects varied in age at the time of the middle adulthood interview, it was important to control for disproportionate risk of developing physical symptoms due to age. Analyses include controls for age at the time of the middle adulthood interview (mean=41.2, SD=3.54, range 32–49).

Welfare as a child, a dichotomous variable, is included as an indicator of childhood poverty to control for economic disadvantage that might affect subsequent risk of pain in middle adulthood. This variable was assessed by the participant’s response to an interview question during young adulthood: "At any time during your childhood, did either of your parents ever receive welfare payments or food stamps?" Responses of "yes" were coded as 1 and others were coded as 0 (50% of the respondents reported welfare in childhood). This measure, rather than an indicator of current economic status, was used as a control, as current economic status might be a consequence of childhood victimization and, therefore, inappropriate as a control variable.

3. Statistical analysis

SPSS version 16 for Windows was used for statistical analyses. Pain symptoms in adulthood were modeled as a function of childhood victimization, controlling for demographic factors (see Control variables, above), using the UNIANOVA procedure, equivalent to analysis of covariance. Levene’s test was used to confirm equality of variances across the subsamples. Models for young and middle adulthood used data on pain symptoms as assessed during the young or middle adulthood period respectively, but the PTSD measure for models predicting pain in either period of adulthood was based on the young adulthood assessment. Separate models were created for any documented childhood victimization (i.e., sexual abuse, physical abuse, or neglect) followed by models for each of the specific types of victimization.

To test for possible mediation through PTSD, we followed steps as established by Baron and Kenney [6], i.e., (a) determine that the exposure measures (i.e., childhood victimization) are correlated with the outcome (i.e., various pain measures); (b) determine that the exposure measures are correlated with the mediator, PTSD; (c) determine that the mediator, PTSD,

affects the outcome when controlling for exposure. If the prior step suggests mediation, we would then assess whether any mediation detected in the prior step is partial or complete.

To test for possible moderation of any childhood victimization/adult pain relationship via PTSD, we contrast coded a term representing all possible combinations of childhood victimization and lifetime PTSD status (i.e., no victimization and no PTSD, victimization only, PTSD only, and both victimization and PTSD), evaluating the magnitude and significance in the difference in pain symptoms between the first group and subsequent groups, enabling easier description of the nature of any statistical interaction. A pure moderator model of PTSD and childhood victimization would be supported in this model, if the subgroup with both PTSD and victimization had higher levels of pain symptoms than groups with neither PTSD or victimization, particularly when PTSD alone or victimization alone did not differ from the subgroup with neither PTSD nor victimization. A more limited moderator model would propose that frequency of pain symptoms were highest in those with both PTSD and victimization, but would be agnostic regarding the difference in symptoms between those with PTSD alone or victimization alone versus the subgroup with neither PTSD nor victimization.

Initial Type I error rate per test was set at $p<.05$. Findings were also Bonferroni corrected for multiple comparisons, with error rate for each group of analyses set at $p<.05$.

4. Results

Comparing the mean number of pain symptoms reported in young and middle adulthood, patterns of change over time varied across the measures: Comparing young versus middle adulthood periods, the total number of pain symptoms decreased (unadjusted $X = 2.36$ (se=.07) to $X = 2.02$ (se=.07), paired $t=4.55$, $p<.001$), as did the number of pain problems (unadjusted $X = 1.98$ (se=.06) to $X = 1.98$ (.06), paired $t=2.02$, $p<.05$). The trend for illness-related pains was toward an increase (unadjusted $X = 1.43$ (se=.05) to $X = 1.54$ (se=.06), paired $t=-1.77$, $p<.10$). The most robust decrease was for medically unexplained pains (unadjusted $X = .52$ (se=.03) to $X = .23$ (se=.02), paired $t=7.65$, $p<.001$.

Given attrition between young and middle adulthood, we first attempted to replicate the young adulthood analyses reported in Raphael et al. [26] for the subset of sample members for whom middle adulthood data were also available. As shown in Table 1, and consistent with the earlier report based on the full sample available in young adulthood, no significant differences were found on any of the pain measures as a function of history of court-documented childhood abuse and/or neglect, even prior to Bonferroni correction.

Table 2 displays adjusted differences in adult pain measures as a function of a history of court-documented childhood abuse/neglect, following up the same sample analyzed in Table 1 into middle adulthood. Prior to adjustment for multiple comparisons, a different pattern emerges for middle compared to young adulthood: With the exception of the measure of the number of unexplained pain symptoms, any form of childhood victimization (i.e., sexual abuse, physical abuse or neglect) significantly predicted ($p<.05$) the number of pain symptoms, the number of pain problems leading to care seeking or activity interference, and the number of pain symptoms ascribed to illness or injury. Results were fairly consistent when examining specific subtypes of child abuse and/or neglect, with significant group differences in the number of pain symptoms as a function of a history of childhood sexual abuse, physical abuse and neglect. For other subtypes of abuse or neglect and pain problems or illness/injury-related pain, results either attained statistical significance ($p<.05$) or showed nonsignificant trends ($p<.10$). Despite a relatively consistent pattern of significant differences as a function of childhood victimization, partial eta squared (η^2) statistics were

uniformly extremely modest, i.e., 1% of variance explained by child abuse/neglect (detail not shown), even for the most reliable effects (i.e., any abuse/neglect and number of pain symptoms). After Bonferroni corrections, there were no significant differences as a function of childhood victimization.

The first step for a test of possible mediation through PTSD (see Statistical Analysis) was accomplished via analyses presented in Table 2, demonstrating that a history of childhood victimization predicts a variety of pain outcome measures in middle adulthood, at least prior to Bonferroni correction. The second step examines the relation between childhood victimization and the potential mediator, PTSD. Of the middle adulthood sample, 28.7% (n=232) met DSM-III-R criteria for lifetime PTSD, as assessed in young adulthood interviews. Of those with lifetime PTSD, 21.6% (n=50) identified the PTSD-related event as involving rape or assault in childhood. Slightly more than half (54.7%, n=127) of those with lifetime PTSD reported current or recent PTSD, with symptoms within the past year. Lifetime PTSD was significantly correlated ($p<.01$) with any type of childhood victimization ($r=.13$), and, specifically, sexual abuse ($r=.15$) and neglect ($r=.14$). Lifetime PTSD was also significantly correlated ($p<.05$) with childhood physical abuse ($r=.12$).

The final two steps for a test of mediation involve the addition of PTSD to the models predicting pain from victimization status and then examining whether childhood abuse or neglect still affects pain when controlling for PTSD; if so, the next step is to determine whether mediation is partial or complete by examining the change in the victimization effect, with versus without the mediator term in the model. Table 3 shows the adjusted differences in pain symptoms as a function of childhood victimization, after controlling for the same set of demographic factors in Table 2, but adding lifetime PTSD status to the model. Comparison of patterns of differences and p-values in Tables 2 and 3 suggest that, at most, PTSD weakly or equivocally mediates the relation between childhood victimization and pain in middle adulthood: although some differences that were significant prior to Bonferroni correction are no longer significant or show a reduction in p-value, overall patterns do not markedly change when adding PTSD to the model.

Failure to find clear evidence of mediation due to PTSD contrasts with the direct effect of PTSD on pain in the demographically-adjusted models (detail not shown), where PTSD explained 3% of the variance in the number of pain symptoms in middle adulthood ($p<.001$). In contrast to the lack of a relation between any type of childhood victimization and unexplained pain in middle adulthood, PTSD predicted 4% of the variance in unexplained pain ($p<.001$).

Tests of moderation involved evaluation of whether consideration of the combined effect of documented childhood victimization and lifetime PTSD uniquely explained pain symptom variation in middle adulthood. Of the 807 participants in middle adulthood, 273 (33.8%) had neither lifetime PTSD nor documented childhood victimization, 76 (9.4%) had victimization but no PTSD, 302 (37.4%) had PTSD only, and 156 (19.3%) had both lifetime PTSD and victimization ($\chi 2=14.59$, $p<.001$, consistent with $r=.13$ detailed above, for the relation between PTSD and victimization).

Table 4 shows differences in mean pain symptoms among these four PTSD by victimization groups, after adjusting for demographic factors. When considering the combined effect of childhood victimization and lifetime PTSD on pain symptoms, a clear pattern emerges: Those with a combination of any childhood victimization or specific victimization as well as lifetime PTSD have a robust increase in number of pain symptoms, pain problems, and pains ascribed to illness or injury. Although some unique patterns emerged for medically unexplained pain, with a suggestion that the combined effect of neglect and PTSD, or any

victimization and PTSD lead to higher rates, these significant ($p<.01$ and $p<.05$ respectively) effects would not survive Bonferroni correction, should all analyses in the table be used to adjust p-values. Furthermore, after considering the combined effect of childhood victimization and PTSD, consideration of only victimization status or PTSD status did not significantly predict pain symptoms in middle adulthood. When examining an overall statistical model, the term representing the combined effect of victimization and PTSD status on pain symptoms predicted a moderate amount of the variance in pain outcomes, for all pain measures other than unexplained pain (i.e., partial η^2 range: .05 – .06 for any victimization, .05 – .06 for sexual abuse, .05– .06 for physical abuse, and .03–.05 for neglect).

5. Discussion

An examination of pain symptoms in middle adulthood as a function of a history of court-documented childhood victimization found that, in contrast to earlier analyses focusing on the same types of symptoms in young adulthood (approximate age 29) [26], statistically significant effects begin to appear in middle adulthood (approximate age 41). However, the magnitude of effect due to childhood victimization still appeared to be modest. We next tested whether this modest relationship can be explained by a process through which childhood victimization leads to PTSD, which in turn increases risk of pain in middle adulthood. Although history of PTSD, as assessed in young adulthood, was associated with both childhood victimization and pain in middle adulthood, it did not appear to mediate the relationship between childhood victimization and adult pain. Thus, the fact that victims of childhood maltreatment are at increased risk for PTSD in adulthood does not seem to be a mechanism through which such individuals are also at increased risk for pain in adulthood.

When we next tested whether those with a history of both PTSD and childhood victimization had increased risk of pain in middle adulthood, results were clear: The combination of a history of childhood abuse or neglect and PTSD led to a marked increase in most types of pain complaints. After accounting for this synergistic effect, the effect of child abuse and neglect alone or PTSD alone did not approach statistical significance.

We had originally anticipated that we might observe pain-related consequences of childhood victimization, as individuals enter a period of greater risk for pain later in life. In fact, with the exception of illness-related events, frequency of pain complaints decreased between young and middle adulthood, with the most robust decrease occurring for medically unexplained pains. The pattern of decrease in unexplained pain symptoms, combined with a trend toward a rise in illness-related pain symptoms, lends some validity to our measure of unexplained pain over time, as continued care seeking in the course of adulthood could lead to a medical label for previously unexplained chronic pain symptoms. Thus, it is of note that patterns for unexplained pain, the type of pain often conjectured to be most influenced by early childhood abuse [24] did not show an overall relationship with childhood victimization status, despite hypotheses that unexplained symptoms are more likely to have psychological causes [16]. Evidence for synergy between childhood victimization and PTSD were less clear when predicting unexplained pain than for other pain outcomes, and relationships that were significant would not survive stringent Bonferroni corrections for multiple comparisons.

The focus of the current analyses involves pain symptoms in middle adulthood. Since the synergy between PTSD and childhood victimization was first empirically addressed in the current investigation but not in our earlier report of this same cohort in young adulthood [26], in supplementary analyses we tested whether an equivalent synergy in prediction of pain symptoms occurred when this same cohort was assessed in young adulthood. In

analyses not detailed here, findings for young adulthood were very similar to the findings for middle adulthood, in which the combination of PTSD and childhood victimization robustly predicted pain symptoms. In models accounting for the interaction, childhood victimization status alone was not a predictor. However, in contrast to middle adulthood findings, PTSD remained a significant predictor of young adulthood pain symptoms. Thus, the interaction between childhood abuse and PTSD appears in both young and middle adulthood.

The concept that PTSD is a marker of a biological propensity toward stress vulnerability rather than a simple reflection of exposure to traumatic events has been advanced by many experts in the area of PTSD. Only a subset of those exposed to extreme stressors are likely to develop PTSD, [39] even when the stressors occur in childhood [11]. The concept of PTSD as a marker of stress vulnerability is supported by prospective research [8] which finds that prior exposure to trauma is a risk factor for PTSD only among the subset of individuals who developed PTSD in response to the earlier trauma. Yehuda and colleagues [46] suggest “the biology of PTSD appears to be one in which there may be a set of pretraumatic biological risk factors that serve to impede the individual's ability to contain the stress response.” (p.1798) Despite the methodological challenge of disentangling severity of trauma exposure from the biology of PTSD risk, an emerging literature has begun to examine the biological and neurophysiological substrates of vulnerability to PTSD (see reviews by [14,20,39,47]).

Despite the fact that our earlier report of young adulthood pain-related consequences of childhood victimization [26] was rated highest in methodological quality among 52 studies discussed in one review of the literature on abuse and medically unexplained symptoms [29], some limitations of the current paper should be noted. First, assessment of “lifetime” PTSD was assessed in young adulthood, not in middle adulthood interviews. Thus, those who might have developed PTSD between young and middle adulthood were considered to be negative for a lifetime history of PTSD. Thus, given this possibility of measurement error, it is striking that PTSD interacted robustly with childhood victimization to predict middle adulthood pain symptoms. Especially since we confirmed that similar synergistic patterns occurred in young adulthood, this suggests that propensity toward stress vulnerability is likely to be manifest by development of PTSD and pain relatively early in life. Nevertheless, since individuals who eventually recover from PTSD may differ from those who do not, failure to measure current PTSD in middle adulthood limits such an exploration.

Although use of official records of child abuse and neglect is an advantage in that it avoids problem inherent in self-report [45], this strategy means that only individuals whose abuse or neglect came to the attention of authorities were classified as being abused or neglected. In addition, the sample is skewed toward the lower end of the socio-economic spectrum, so that results may not generalize to other socioeconomic groups. The sample also represents individuals who grew up in the late 1960s and early 1970s in the Midwestern part of the United States, and findings may not generalize to individuals from other generations or geographic regions. Additionally, cases of child abuse and neglect occurred before age 12, so that findings may not generalize to abuse or neglect in adolescence.

Although there was no evidence of differential loss to follow-up as a function of victimization status or key demographic measures, attrition over the course of 30 years of follow-up limits to the extent to which the results can be confidently generalized to the original cohort of 1575 individuals. Concern about potential sampling bias is somewhat lessened through analyses showing that relationships between victimization and pain outcomes in early adulthood were the same for the sample followed into middle adulthood as for the larger sample interviewed in early adulthood, as reported in an earlier

publication[26].It is worth noting that those with documented histories of childhood abuse and/or neglect in the 1960s and early 1970s were unlikely to receive the kinds of therapeutic interventions provided today when such victimization comes to the attention of authorities. Many of the children in the current study were removed from their homes, leading to potential exacerbation rather than buffering of the consequences of victimization. Thus, childhood victimization in this cohort was likely to lead to a cascade of major life changes. In this context, the fact that pain-related outcomes were only found for the subset of adulthoods that also had PTSD is striking.

Overall, these findings support our earlier contentions [22–24,31] that, in routine clinical assessments, pain patients are not likely to be well served by a broad inquiry about a past history of childhood abuse and neglect. Rather, these data support a view that clinical assessment should focus on signs and symptoms of PTSD. Assessment of abuse histories may prove to be relevant for the subset of pain patients presenting with PTSD, if there is an indication that childhood experiences were the triggering event for PTSD.

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Table 1

Adjusted Mean Pain Symptoms in Early adulthood by Documented Childhood Victimization Status, Among the Sample Followed into Middle Adulthood (Controlling for age, sex, race, and welfare status as a child)

Pain Measure:	Any Documented Abuse or Neglect (n = 458)		Control (n =349)		Documented Sexual Abuse (n = 61)		Control (n = 349)		Documented Physical Abuse (n =78)		Control (n =349)		Documented Neglect (n = 370)		Control (n = 349)	
	M	(se)	M	(se)	M	(se)	M	(se)	M	(se)	M	(se)	M	(se)	M	(se)
# Pain Symptoms	2.38	(.09)	2.24	(.10)	2.29	(.28)	2.18	(.11)	2.19	(.26)	2.17	(.11)	2.23	(.11)	2.15	(.11)
# Pain Problems	2.00	(.08)	1.88	(.09)	1.96	(.26)	1.83	(.10)	1.86	(.23)	1.82	(.10)	1.94	(.10)	1.80	(.10.)
# Pains Attributed to Illness/Injury	1.47	(.07)	1.35	(.08)	1.33	(.21)	1.34	(.08)	1.43	(.19)	1.33	(.08)	1.49	(.09)	1.31	(.09)
# Unexplained Pains	0.50	(.04)	0.50	(.04)	0.60	(.14)	0.48	(.06)	0.41	(.12)	0.48	(.05)	0.41	(.05)	0.47	(.05)

Note: No significant group differences or trends, even prior to Bonferroni correction

Table 2

Adjusted Mean Pain Symptoms in Middle Adulthood by Documented Childhood Victimization Status (Controlling for age, sex, race, and welfare status as a child)

Pain Measure:	**Any Documented Abuse or Neglect**		**Control**		**Documented Sexual Abuse**		**Control**		**Documented Physical Abuse**		**Control**		**Documented Neglect**		**Control**	
	(n = 458)		**(n =349)**		**(n = 61)**		**(n = 349)**		**(n =78)**		**(n =349)**		**(n = 370)**		**(n = 349)**	
	M	**(se)**	**M**	**(se)**	**M**	**(se)**	**M**	**(se)**	**M**	**(se)**	**M**	**(se)**	**M**	**(se)**	**M**	**(se)**
# Pain Symptoms	2.24	(.10)	1.77	(.11)**	2.39	(.25)	1.78	(.10)*	2.33	(.23)	1.75	(.10) *	2.19	(.11)	1.75	(.10) **
# Pain Problems	1.98	(.09)	1.66	(.10)**	2.26	(.24)	1.65	(.10)*	2.05	(.22)	1.62	(.10)′	1.92	(.10)	1 .63	(.10)*
# Pains Attributed to Illness/Injury	1.66	(.08)	1.40	(.09)*	1.89	(.22)	1.40	(.09)*	1.78	(.20)	1.37	(.09)′	1.61	(.09)	1.37	(.09)′
# Unexplained Pains	0.26	(.03)	0.21	(.04)	0.32	(.08)	0.21	(.03)	0.25	(.07)	0.21	(.03)	0.26	(.04)	0.21	(.04)

* p<.05

** p<.01

t p<.10

PRIOR TO BONFERRONI CORRECTION

No significant differences after Bonferroni correction

Table 3

Adjusted Mean Pain Symptoms in Middle Adulthood by Documented Childhood Victimization Status Controlling for age, sex, race, and welfare status as a child *and lifetime PTSD status*

Pain Measure:	**Any Documented Abuse or Neglect**		**Control**		**Documented Sexual Abuse**		**Control**		**Documented Physical Abuse**		**Control**		**Documented Neglect**		**Control**	
	(n = 458)		**(n =349)**		**(n = 61)**		**(n = 349)**		**(n =78)**		**(n =349)**		**(n = 370)**		**(n = 349)**	
	M	**(se)**	**M**	**(se)**	**M**	**(se)**	**M**	**(se)**	**M**	**(se)**	**M**	**(se)**	**M**	**(se)**	**M**	**(se)**
# Pain Symptoms	2.20	(.10)	1.82	(.11)*	2.33	(.25)	1.79	(.10)*	2.26	(.23)	1.76	(.10) **	2.15	(.11)	1.79	(.10) *
# Pain Problems	1.93	(.09)	1.71	(.10)*t*	2.20	(.24)	1.66	(.10)*	1.99	(.22)	1.63	(.10)	1.87	(.10)	1.67	(.10)
# Pains Attributed to Illness/Injury	1.63	(.08)	1.43	(.09)	1.84	(.22)	1.40	(.09)*t*	1.73	(.20)	1.37	(.09)	1.58	(.09)	1.40	(.09)
# Unexplained Pains	0.26	(.03)	0.22	(.04)	0.31	(.08)	0.21	(.03)	0.24	(.07)	0.21	(.03)	0.25	(.04)	0.22	(.04)

* p<.05

** p<.01

t p<.10

PRIOR TO BONFERRONI CORRECTION

No significant differences after Bonferroni correction

Table 4

Difference in Adjusted Mean Pain Symptoms in Middle Adulthood Broken Down By the Combined Effect of Documented Childhood Victimization Status and Lifetime PTSD status

Pain Measure:	No Documented Abuse or Neglect Vs.			No Documented Sexual Abuse Vs			No Documented Physical Abuse Vs			No Documented Neglect Vs		
	Abuse/neg only	PTSD only	Both	Sexual Abuse only	PTSD only	Both	Phys. Abuse only	PTSD only	Both	Neglect only	PTSD only	Both
# Pain Symptoms	.34	.15	**1.32*****	.34	.08	**1.79*****	.35	.05	**1.75*****	.36	.19	**1.13*****
# Pain Problems	.34	−.01	**1.19*****	.33	.02	**1.87*****	.33	−.17	**1.73*****	.35	.03	**.98*****
# Pains Attributed to Illness/Injury	.21	−.02	**.98*****	.21	−.14	**1.71*****	.20	−.16	**1.58*****	.22	.03	**.76*****
# Unexplained Pains	.14	.04	.18*	.13	.21*	.02	.14^{t}	.04 ^{t}	.12	.14	.02	.21**

* p<.05

** p<.01

*** p<.001

^{t} p<.10

PRIOR TO BONFERRONI CORRECTION

Bold signifies significant differences after Bonferroni correction